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Shipping Fever of Cattle

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North Central Regional Project (NCR-29) on Shipping Fever of Cattle

FOREWORD

This publication represents the results of research conducted by the North Central Regional Technical Committee (NC-44 and NCR-29) on shipping fever of cattle from 1957 to 1964. The objectives of this project were:

1. To study the clinical characteristics of the disease.
2. To determine the etiological agent or agents.
3. To study the pathogenesis and the epizootiology of the syndrome.
4. To study and evolve methods whereby shipping fever can be controlled and prevented.

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Acknowledgment is given to all former members and members of the NC-44 Technical Committee on Shipping Fever and their teams who contributed valuable information included in this bulletin.

To the Memory of
Dr. Harvey H. Hoyt
1915-1964

Dr. Hoyt was one of the originators of the North Central Technical Committee on Shipping Fever of Cattle (NC-44) and his continued efforts contributed to its success. He was chairman of the Technical Committee for 1957-1958.

Dr. Hoyt was Associate Dean of Veterinary Medicine at the University of Minnesota and Head of the Department of Medicine and Clinics from 1954 until his death on December 18, 1964.

Dr. Hoyt was a member of the Publication Committee which made this bulletin possible. Therefore, this bulletin is dedicated to his memory.

Shipping Fever of Cattle

A. H. HAMDY, C. C. MORRILL, AND H. H. HOYT

INTRODUCTION AND HISTORICAL REVIEW

Shipping fever of cattle is a complex respiratory disease causing great losses to the livestock industry in many ways. These may include: poor weight gain, loss of meat production due to stunting, lengthening of the feeding period, and death of the animal. The Agricultural Research Service, U. S. D. A., estimates that the annual economic loss to the livestock industry from this disease amounts to more than $25 million. This loss does not include the cost of attempted prevention or treatment of sick animals.

Shipping fever was probably recognized and first described about 1878 in Germany, where it caused considerable losses among stags and wild hogs (51). A short time later, it was reported in cattle and other animals. The fact that the disease was infectious was recognized in 1881.

Louis Pasteur described the bacterium that causes fowl cholera in 1880. Later, it was learned that fowl cholera organisms could not be differentiated culturally from organisms of cattle pneumonia. The generic name *Pasteurella* was proposed for this organism in honor of Pasteur. The name pasteurellosis was proposed for the disease since the bacterium was believed to be the principal or exciting cause.

The disease has been referred to as transit fever, stockyard disease, stockyard fever, pneumonia complex, hemorrhagic septicemia, cattle fever, shipping fever complex, and pasteurellosis.

Because different species of animals had diseases associated with *Pasteurella* infection, some scientists believed these conditions were caused by the same organism. Others thought that the various animals were infected by closely related bacteria belonging to the same genus but different species. In 1939, it was proposed that most of these organisms be regarded as belonging to a single species and that it be called *Pasteurella multocida* (52). Meanwhile, another species known as *Pasteurella hemolytica* had been encountered with considerable frequency in the shipping fever complex (53).

Certain observations have led many scientists to doubt that the disease is due solely to bacteria of the *Pasteurella* group. These include:

1. Difficulty in producing the condition experimentally in many animals solely by inoculation with the bacteria.
2. Isolation of the bacteria from apparently normal animals.
3. The tendency of the disease to occur following stress of some kind, such as shipping, overcrowding, weaning, exhaustion, and change in weather conditions.
4. Different results and opinions relative to efficacy of the immunization procedure with either *Pasteurella* bacterins or *Pasteurella* immune sera.

CAUSES

Agents Associated with Shipping Fever

Shipping fever of cattle is a complex disease. It can best be described as a multiple infection due to the interaction of viral and bacterial multiplication along with stress.

Extensive work has been done to determine the causative agents (1-12, 14-39, 43, 44). In the past, some investigators suggested that *Pasteurella* organisms were the primary cause of the disease. However, they were unable to induce the disease experimentally in calves infected with various strains of *Pasteurella* organisms alone (22).

Strains of pleuropneumonia-like organisms (PPLO) have been isolated from the respiratory tract of calves having clinical signs of shipping fever (23). However, these organisms, when inoculated in calves with or without *Pasteurella,* produced no signs of the disease.

Some investigators have postulated over the years that an epizootic viral infection may alter the respiratory tract, allowing secondary bacteria to produce clinical signs of shipping fever.

This theory of viral causation has gained support since 1959 when Reisinger and his associates at the National Animal Disease Laboratory reported isolation of a virus from cattle with clinical signs of shipping fever (37). This viral agent was found to be a myxovirus known as parainfluenza-3.

Since 1959, viral agents of similar characteristics have been isolated from many areas of the North Central Region, as well as other parts of the world (1, 27, 37, 43). A number of other viral agents have been isolated, including some of the psittacosis-lymphogranuloma group (PLV), infectious bovine rhinotracheitis (IBR) virus, and bovine enteroviruses.

Transmission Studies

Experimental exposures in calves were made to study the interrelationships of some of these agents mentioned previously. Alterations in environmental temperatures were applied as physical stress (31, 33). The agents were inoculated singly and in different combinations to evaluate their role in shipping fever in calves under controlled conditions.

The results obtained from this study indicated that *Pasteurella* organisms, PPLO, or physical stress used separately or in combination were not capable of producing the shipping fever syndrome (31). Thus, the data indicated that none of these agents was the primary cause of shipping fever.

Pasteurella organisms could be isolated from apparently normal calves and from clinical cases of shipping fever (2). It is suggested that the organisms may multiply and invade the tissues when resistance of the nasal mucous membranes is lowered by some kind of stress. It was noted that the severity of pneumonic lesions was related to the frequency of isolation of *Pasteurella* organisms from the nasal passages. This may indicate that *Pasteurella* organisms are responsible for at least some of the lesions produced in shipping fever.

Clinical signs similar to those observed in shipping fever were induced experimentally in physically stressed calves exposed to a combination of myxovirus parainfluenza-3 and *Pasteurella* organisms (Table 1).

TABLE 1.—Results of Exposing Calves to Myxovirus Parainfluenza-3, PPLO, and Pasteurella in Different Combinations.

Agent	Clinical Observations	Pneumonia
MP-3	Off feed and leukopenia	0/4
P	Negative	0/2
PPLO	Negative	0/2
S	Negative	0/2
MP-3 + S	Fever and leukopenia	0/2
MP-3 + P	Fever, cough, off feed, and nasal discharge	2/2
MP-3 + PPLO	Negative	0/2
MP-3 + P + S	Fever, off feed, cough, nasal discharge, and leukopenia	4/4
MP-3 + PPLO + S	Fever and cough	0/2
MP-3 + PPLO + P	Fever, cough, off feed, and leukopenia	1/2
MP-3 + PPLO + P + S	Fever, cough, off feed, and depression	2/2
Control	Negative	0/4

MP-3 = myxovirus parainfluenza-3
P = Pasteurella organisms
S = stress

TABLE 2.—Effect of Exposing Calves to a Combination of Agents Associated with Shipping Fever.

Agent	Clinical Observations	Pneumonia
MP-3+P+S	Fever, cough, off feed, nasal discharge, and leukopenia	2/2
IBR+P+S	Fever, cough, off feed, and leukopenia	2/2
PLV+P+S	Fever, depression, off feed, and leukopenia	0/2
MP-3+IBR+S	Fever, cough, and depression	0/2
MP-3+IBR+P+S	Fever, off feed, nasal discharge, and leukopenia	1/2
MP-3+PLV+S	Fever, cough, and depression	2/2
MP-3+PLV+P+S	Fever, cough, off feed, depression, and leukopenia	2/2
Control	Negative	0/4

IBR = infectious bovine rhinotracheitis virus
PLV = psittacosis-lymphogranuloma venereum virus

In this experiment, the inability of myxovirus parainfluenza-3 to induce shipping fever by itself indicated that it also was not the sole cause of shipping fever. This investigation suggested that neither stress nor a single infectious agent was solely responsible for producing shipping fever, but that multiple factors are involved in pathogenesis of the syndrome.

Some evidence has been presented indicating that myxovirus parainfluenza-3 may, in acute outbreaks, become sufficiently virulent to produce a syndrome in experimental calves, somewhat similar to that observed in natural outbreaks (9). However, bacterial infections superimposed upon viral infections could accentuate the signs and lesions.

Shipping fever was transmitted by contact under field conditions from calves infected with *Pasteurella multocida, P. hemolytica,* and myxovirus parainfluenza-3. The susceptible contact calves developed acute signs of shipping fever. Calves artificially exposed to the disease agents also developed clinical signs of shipping fever but in a milder form (32, 33).

The effect of exposing calves to a combination of one or two viral agents and stress with or without *Pasteurella* organisms was also studied (Tables 1 and 2). The results of this study point to viruses (myxovirus parainfluenza-3, IBR, or PLV—singly or in combination) as the causative factor or factors in naturally occurring cases of shipping fever (33).

More severe signs of the disease were observed in calves exposed to combinations of *Pasteurella*, two viral agents, and physical stress than in

those inoculated without *Pasteurella.* It seems apparent that these agents, along with stress, may combine to produce the disease.

The multiplicity of viral infection excites speculation that virus or viruses may exist as a part of the microflora of the respiratory tract and may tend to cause disease under appropriate conditions. It has not been determined whether latent viruses are activated.

SPREAD

Results of extensive serological studies indicated widespread distribution of bovine myxovirus parainfluenza-3. A survey conducted in Nebraska indicated that 86 percent of 2,843 cattle tested had significant antibody titers to myxovirus parainfluenza-3 (12). The survey also revealed that a high percentage of beef as well as dairy cattle had been exposed to this virus.

In Illinois, serological studies in 11 different groups of feeder cattle indicated that 69 percent of the cattle tested had antibodies against myxovirus parainfluenza-3 (1). Other animals, such as sheep and deer, reacted to the virus, indicating possibility of the prevalence of myxovirus parainfluenza-3 in animals other than cattle.

The effects of shipment on body temperatures and white blood cells of cattle were studied after shipment and arrival at feedlots and/or during naturally occurring outbreaks of shipping fever in Ohio and Illinois. Reduction in white blood cell count was detected in some animals. The averages and ranges of daily temperature and white blood cell count are summarized in Table 3. In some instances, the number of white blood cells increased and the counts remained high for a few days. In a number of calves showing signs of shipping fever, this increase in white blood cells was due to neutrophilia.

Stress Factors

Predisposing factors other than infectious agents have been investigated in Wisconsin and other states (35-37, 40-47). The increase of reported cases of shipping fever in recent years has closely paralleled the increase of moving and shipping cattle from range to feedlot.

The most striking factor of this disease is the severe stress to which cattle are subjected after leaving the range (21). Some of these stress factors are: weaning, changes in environmental condition, improper handling, excitement, exhaustion, irregular feeding and watering, branding, overcrowding, irritation of the mucous membranes of the respiratory tract, and exposure to pathogenic organisms. Some of these factors or others may help to lower the resistance of the animals and render them more susceptible to the infection.

TABLE 3.—Body Temperature and Total Leukocyte Count—Daily Averages and Ranges in Western Cattle after Arrival at Feedlots.

No. Tested	Days After Arrival	Temperature Average F.	Temp. Range	WBC Average	WBC Range
12	4	103.1	101.0-105.5	10,133	5,900-14,550
26	5	103.0	101.6-104.8	8,680	3,995-12,554
12	6	102.7	101.2-104.4	10,446	7,500-14,500
12	7	103.1	102.0-104.4	9,400	6,950-11,400
14	8	103.5	101.0-107.2	8,991	6,307-13,034
14	9	103.4	101.8-104.2	8,961	6,491-10,722
26	10	103.2	100.8-106.6	8,884	4,423-14,800
26	11	103.4	101.2-108.6	9,107	5,151-16,500
26	12	103.1	101.8-106.4	10,040	3,344-15,200
12	13	103.0	101.6-104.8	11,608	7,150-17,650
12	14	103.2	102.4-104.8	12,313	7,600-23,900
14	15	104.3	102.0-107.0	7,880	2,942-13,733
14	16	104.2	101.4-106.2	6,693	1,951- 9,881
14	17	103.4	102.0-105.0	7,104	2,171-12,347
14	18	103.6	102.0-105.8	6,762	1,973-11,515
14	19	103.4	101.5-105.0	7,065	2,549-10,742

WBC = white blood cells (leukocytes) per cu. mm. of blood

There is some evidence that handling and shipping of calves produce a measurable disturbance in adrenocortical activity. It was indicated that calves lack the adaptability of cows to adjust to their environment.

Calves weaned just before being shipped seem to be the most susceptible to the infection. However, resistance seems to increase with age of the animals. Undernourished and highly parasitized animals are usually more seriously affected. Deficiencies of vitamins, such as vitamin A, could also be expected to reduce the resistance of the animals to respiratory infection. However, other workers reported that the use of vitamin A with or without antibiotics did not lower the incidence of shipping fever.

SYMPTOMS AND DIAGNOSIS

Shipping fever is primarily a respiratory syndrome of cattle that varies from an inapparent disease to a rapidly-developing fatal pneumonia. Signs of the disease may appear within 2 to 21 days (mostly 5 to 14 days) of arrival at the feedlots.

The first signs of the disease are a tired appearance and reduced appetite. The affected animal may also show signs of depression, watery

to mucopurulent nasal discharge, increase in body temperature (sometimes reaching as high as 108.8°F.), an occasional soft or hacking cough, rapid breathing, and loss of body weight. The death rate varies from 0 to 10 percent, depending upon the severity of the infection. Chronic pneumonia may result in some cases.

DIFFERENTIAL DIAGNOSIS

Diagnosis of shipping fever presents certain difficulties. Cattlemen, research workers, and practicing veterinarians often differ in their diagnosis of this disease (25). Some people suggest that the shipping fever syndrome represents several conditions that vary from one area to another.

Efforts should be made to differentiate between shipping fever and several other diseases that might confuse the diagnostic picture. Such diseases include IBR, mucosal disease-virus diarrhea complex, and pneumonias of other origin. Tissue culture, bacteriological, and serological techniques may be required to differentiate shipping fever from other similar diseases.

Careful observation of the affected animals, laboratory studies, and postmortem examinations are sometimes necessary to arrive at a correct diagnosis. Shipping fever is indicated when signs of respiratory illness, accompanied by fever and poor appetite, are observed in cattle that have been shipped within 2 to 21 days.

On postmortem examination, the affected animals may show pathologic changes confined chiefly to the respiratory organs. The character and extent of these changes depend on length of the illness, severity of the infection, and complicating secondary infections.

Bronchopneumonia with congestion of the lung occurs in the early stage of the disease. Some animals may have hemorrhage, fibrinous exudate, as well as inflammation of the lining of the thoracic wall (pleuritis) with or without pericarditis and pneumonia. The air spaces of the lung (alveoli) may contain fibrin and blood. Edema may be noticed in some areas of the lung.

In later stages of the disease, secondary bacteria may invade the tissues and bloodstream, producing bacteremia. The latter may be indicated by small hemorrhages on various organs.

Lung abscesses may develop in some animals. The thoracic lymph nodes are swollen and congested. Changes in organs or tissues outside the respiratory system are probably due to toxemia, bacteremia, or both.

PUBLIC HEALTH ASPECTS

Recent investigations of common respiratory diseases in children and adults have emphasized the importance of myxovirus parainfluenza types 1, 2, and 3 in acute respiratory infections (48, 49). Biological and antigenic characteristics of the bovine strain of myxovirus parainfluenza-3 appear closely related to the human strain. These similarities suggest the possibility of some type of host relationship between these agents.

An outbreak of myxovirus parainfluenza-3 infection was reported in a group of orphanage children in 1963 (50). The clinical signs were very mild, with continuous respiratory illness. Cough, fever, nasal discharge, and pulmonary involvement increased during this outbreak. Myxovirus parainfluenza-3 was isolated from 25 percent of the children observed.

In Ohio, serologic studies of persons who had contact with cattle indicated that some had antibody titers against influenza virus types A, B, or myxovirus parainfluenza-3.

PREVENTION AND CONTROL

Numerous studies have been made in the North Central Region to prevent and control shipping fever (3-5, 7, 9, 14, 19, 29, 30, 32, 34, 36).

For many years, efforts to prevent shipping fever were confined chiefly to using *Pasteurella* bacterin (shipping fever bacterin) and hemorrhagic septicemia antiserum. Use of such biological products has proved of little or no value in prevention of the disease. In fact, losses were sometimes higher in inoculated than in uninoculated animals. Many research workers in this country failed to observe any beneficial results from the use of immune serum.

Several medications have been used for the prevention and/or treatment of shipping fever in the field (15). Procaine penicillin, dihydrostreptomycin, hemorrhagic septicemia bacterin, hemorrhagic septicemia antiserum, tranquilizers, and IBR vaccine, singly or in various combinations, were used in a total of 14,034 calves (Table 4). Results from this study indicated that none of these products prevented the occurrence of shipping fever. However, some products appeared to have a beneficial effect. The study indicated that management is a primary factor in prevention of the disease.

In another study, penicillin injection and hemorrhagic septicemia antiserum, singly and in combination, were used in three groups of calves (19). The authors reported that no final conclusions were reached.

However, hemorrhagic septicemia antiserum, when used prophylactically in cattle following shipment, had little or no value in controlling shipping fever (Table 5).

In spite of the questionable value of the bacterin or serum, millions of doses are used every year.

Some antibiotics were found effective in reducing the incidence of shipping fever when injected into animals before shipment and soon after arrival at feedlots. However, the true value of these drugs is not clear. Tranquilizers were found to be worthless in preventing the disease. So treatment with these drugs is a useless expenditure.

TABLE 4.—Comparison of Various Medications in the Control of Shipping Fever.

Treatment	Total Lots	Total Calves	Percent Receiving Medication before Developing Signs Lots	Percent Receiving Medication before Developing Signs Calves	Percent Showing Signs at 3 Weeks Lots	Percent Showing Signs at 3 Weeks Calves
Control	394	2473	24	16	10	9.4
Penicillin I	152	4114	19	11	45	4.6
Penicillin II	109	2471	38	8.2	17	4.4
Pen. III—Streptomycin	14	328	14	9	50	3.3
Tranquilizer	57	1874	11	7.9	15.8	2
Hem. sep. bacterin (x2)	17	265	66	88	35	9.7
Pen. II—Hem. sep. bacterin	3	196	100	100	33.3	2.7
Hem. sep. serum (130 ml.)	5	163	10	0	20	1.5
IBR vaccine	24	1417	84	85	35	3.1
Hem. sep. serum (30 ml.) bact.	60	733	66	33	20	2.1

Pen. = penicillin

Hem. sep. = hemorrhagic septicemia

TABLE 5.—Effectiveness of Various Prophylactic Treatments for Control of Shipping Fever.

Prophylactic Treatment at Time of Shipping	Total Animals	Animals Requiring Treatment No.	Animals Requiring Treatment Percent	Death Due to SF
Penicillin only	73	15	20	2
Serum only	73	21	29	3
Penicillin and serum	33	5	15	1
Control	69	30	4.3	3
Total	248	71	29	9

TABLE 6.—Summary of Clinical Observations and Virus Isolations in Control and Vaccinated Calves.

Treatment	No. of Calves	Calves with Signs of Shipping Fever		No. of Virus Isolations
		No.	Percent	
Vaccinates	43	9	20.9	5
Controls	44	5	11.4	3
Total	87	14	16.0	9

Research is now in progress in the North Central Region to develop new vaccines and to evaluate their effectiveness. Myxovirus parainfluenza-3 vaccine was used in western calves prior to shipment (3). Signs of the disease were observed in both the vaccinates and the controls (Table 6). Injection of a myxovirus parainfluenza-3 vaccine in beef calves 1 month before weaning had no effect on incidence of the disease (7).

Since the disease was successfully transmitted in stressed calves exposed to a combination of myxovirus parainfluenza-3 and *Pasteurella* organisms, attempts were made to immunize calves against these agents under experimental as well as natural conditions (34, 36). Data in Tables 7 and 8 show that a substantial degree of protection against the disease appeared to be produced in calves vaccinated with preparations containing myxovirus parainfluenza-3; *Past. hemolytica* and *Past. multocida* types II, III, and IV; or myxovirus parainfluenza-3 and IBR virus. However, the same preparations gave no protection against shipping fever under the field study.

In studying the cause of the disease complex, different strains of the virus were recovered. These strains shared common characteristics such

TABLE 7.—Effect of Vaccination against Shipping Fever and Challenge under Experimental Conditions.

Vaccine Used	Calves Used	Calves Resisting Challenge*	
		No.	Percent
MP–3, **Past. mult.** type I	4	1	25
MP–3, **P. hemol., P. mult.** types II, III, IV	4	3	75
MP–3, IBR	4	3	75
Controls	4	0	0
Total	16	7	43.7

*Absence of pneumonia and any signs of respiratory infection following challenge with homologous agents.

as cytopathic effect in bovine kidney cell culture; hemagglutination of bovine, human O, guinea pig, and porcine red blood cells; ability to form plaques in cell cultures; and sensitivity to ether or formalin (35). However, these strains varied in their hemagglutinating activity as well as their heat inactivation at 56° C. (Table 9). Thus, the existence of different strains of the virus was demonstrated.

It is evident that more work is needed to modify the vaccine preparation to protect cattle against naturally occurring cases of shipping fever.

TABLE 8.—Effect of Vaccination against Shipping Fever under Field Conditions.

Type Vaccine Used	No. Calves	Clinical Signs of Shipping Fever No.	Percent
MP–3, **Pasteurella** spp.*	30	5	16.6
MP–3, **Past. mult.** type I	20	4	20.0
MP–3, IBR	20	4	20.0
Controls	95	14	14.7
MP–3, **Past.****, IBR	55	4	7.2
Controls	80	12	15.0
Total	300	43	14.3

*Pasteurella spp. = **P. hemolytica, P. multocida** types II, III, and IV.

Past. = **P. multocida type II and **P. hemolytica.**

TABLE 9.—Hemagglutination (HA) Titers against 13 Myxovirus Parainfluenza-3 Viral Isolates with Erythrocytes from Six Different Species.

Virus Strain	Erythrocytes					
	Avian	Bovine	Ovine	Porcine	Guinea Pig	Human O
19	20	40*	20	80	320	20
41	Neg.	80	Neg.	320	320	160
43	Neg.	20	Neg.	80	160	20
54	Neg.	20	Neg.	320	640	40
57	10	40	20	80	80	20
59	20	20	20	40	40	40
77	10	40	10	40	40	20
78	20	40	10	80	80	20
85	160	640	80	1280	1280	40
118	10	40	20	320	320	40
133	40	40	40	320	320	40
142	Neg.	20	10	320	320	40
SF-4	Neg.	40	40	80	160	80

*Reciprocal HA titer.

FUTURE PLANS

Further work needs to be done along the following lines:

- Continue the search for other causative agents, especially viral and bacterial.
- Attempt to characterize these agents and determine their primary importance, including study of their pathogenicity in germ-free animals.
- Develop a national typing center or centers for pathogenic viruses as a service to all workers doing research on human and animal viruses.
- Critically evaluate stress, management practices, and nutritional factors.
- Determine more suitable methods of handling cattle to reduce exposure during shipment.
- Continue investigations on control and prevention of shipping fever by active or passive immunization.

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